UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

REGION VII
901 NORTH 5TH STREET
KANSAS CITY, KANSAS 66101

DEC 0 3 2007

MEMORANDUM

SUBJECT: Review of Draft Human Health Risk Assessment
Herculaneum Lead Smelter Site
Herculaneum, Missouri

FROM: Mike Beringer, Toxicologist
ENSV/EAMB

TO: Bruce Morrison, Remedial Project Manager
SUPR/FFSE

As requested, we have reviewed the draft "Community Risk Assessment" for the Herculaneum Lead Smelter Site, located in Herculaneum, Missouri. While the majority of our previous comments on the RAGS Part D Interim Deliverables Report and Bioavailability Memorandum were adequately addressed, several issues still remain that should be addressed in the final human health risk assessment. We recommend that Doe Run follow our comments explicitly or seek clarification before revising the document, to ensure that the next draft is the final version.

If you have any questions regarding the attached comments, please let me know.

Attachment

Comments on the Draft Human Health Risk Assessment
Herculaneum Lead Smelter Site
Herculaneum, Missouri

General Comments

1. Additional explanation and detail would greatly improve the overall transparency of the Human Health Risk Assessment (HHRA). The HHRA should be written so as to allow readers to understand all of the steps, logic, key assumptions, limitations, and decisions in the risk assessment. For example, the introduction to several sections should briefly explain the concepts to be discussed in that section to ensure the public can fully understand how the potential health risks have been characterized.

Specific Comments

1. Section 1.3 (p. 2) The four parts of a risk assessment discussed in this section should match those outlined in the "Risk Assessment Guidance for Superfund, Part A," (EPA, 1989), as should the general outline of the document (see Exhibit 9-1).

2. Section 2.2 (p. 7) (a) This section should also reference and briefly discuss a figure of the conceptual site model that represents the linkages among contaminant sources, release mechanisms, exposure pathways and routes, and receptors. (b) For all residential exposure areas, the risk assessment should assume that child and adult residents live in a single home for 6 and 24 years, respectively, for a total exposure duration of 30 years.

3. Section 3.1 (p. 13) As requested previously by Region 7, the risk assessment must include additional details on soil sample collection (e.g., sieve size, etc). In addition, the discussion should reference Section 3.5 concerning data useability.

4. Section 3.1.2 (p. 15) The regression analysis should be revised to evaluate the correlation between the XRF results as the independent variable "x" and the laboratory results as the dependent variable "y." In addition, all data used in the regression analysis should be provided, as well as the statistical output, including 95% confidence intervals for the regression equation parameters. Doe Run should use these results to determine whether a "correction factor" is warranted to adjust the XRF results to yield a laboratory estimate before calculating an exposure point concentration for lead. This determination should be based on the regression equation for soil concentrations less than 2000 mg/kg because the draft HHRA shows that the correlation varies with concentration and the XRF instrument slightly underestimates laboratory concentrations less than 2000 mg/kg.

5. Section 3.2 (p. 16) As requested in previous comments by Region 7, the risk assessment must include additional details on interior dust data sample collection, including sampling methodology, sieve size, presence of lead-based paint, etc.

6. Section 3.2 (p. 17) The risk assessment states that EPA indoor dust data could not be used because property addresses were unavailable. Region 7 will provide these data for inclusion in the risk assessment. Doe Run should also use these data to discuss whether recontamination of home interiors is occurring from the lead smelter.

7. Section 3.3.1 (p. 17) It is unclear why the air monitoring data collected by the Missouri Department of Natural Resources (MDNR) were not included in the risk assessment. These data should be added to the risk assessment and evaluated for potential use.

8. Section 3.3.1 (p. 18) Doe Run should use the latest air monitoring data which reflect current conditions at the site, as opposed to relying on data collected in 2003.

9. Section 3.4 (p. 22) As requested previously by Region 7, the laboratory detection limits should be provided for each compound listed in Table 5.

10. Section 3.5 (p. 22) While the Risk Assessment Guidance for Superfund (RAGS) Part D Data Useability Worksheets were added to the HHRA, this section must also briefly discuss how the evaluation criteria in EPA's "Guidance on Data Useability in Risk Assessment" have been adequately satisfied for each media.

11. Section 5.1 (p. 25) As a point of clarification, ProUCL calculates several estimates of the upper confidence limit (UCL) on the mean concentration and not just the 95% UCL. As previously requested, the risk assessment should provide documentation of the exposure point concentration recommendations generated by ProUCL in a separate appendix.

12. Section 6.3.2 (p. 34) Doe Run should revise the next to last sentence to state "...used to predict BLLs for the child resident, as well as 5 and 6 year old children who attend Taylor School."

13. Section 7.1 (p. 35) (a) The HHRA should briefly define a reasonable maximum exposure (RME) and central tendency exposure (CTE) scenarios. The text should also indicate that the Herculaneum risk assessment only addresses the RME scenario for non-lead Constituents of Potential Concern (COPC)s. (b) Doe Run should delete footnote 9 because the "Exposure Factors Handbook" (EPA, 1997) indicates that a soil ingestion rate of 200 mg/day is appropriate for RME scenarios. In addition, EPA's "Child-Specific Exposure Factors Handbook" (EPA, 2002) supports the use of 200 mg/day as a conservative mean estimate.

14. Section 7.1 (p. 36) As previously requested by Region 7, the risk assessment should use a soil adherence factor of 0.2 mg/cm^2 for elementary school children. This value is based on

children playing in wet soil and is consistent with RAGS Part E (EPA, 2004) which recommends using a high-end soil contact activity with a central tendency weighted adherence factor for that activity. The text and all tables should be revised accordingly.

15. Section 7.2 (p. 37) (a) In July 2000, EPA determined that a specific *in vitro* bioaccessibility assay (IVBA) is considered an appropriate regulatory methodology for estimating the relative bioavailability of lead for quantitative use in site-specific risk assessments (see www.epa.gov/superfund/health/contaminants/bioavailability/transmemo_rel_bio.pdf). The text should be changed to reflect the Agency's new policy, but the risk assessment should continue to rely on the *in vivo* bioavailability results for predicting blood lead levels. (b) Doe Run has repeatedly told Region 7 that the samples collected for the Casteel *et al.* (2001) bioavailability study were not representative of the site for unspecified reasons. Region 7 was not present when the samples were collected and was also not notified of the sampling event. The bioavailability report, dated June 2001, is stamped "Draft" and to EPA's knowledge has not been finalized. Thus, Doe Run must acknowledge there are data quality issues associated with this study. As a result, there is significant uncertainty with the study and in comparing the results to more recent bioavailability data.

16. Section 7.2.3 (p. 40) Doe Run should provide in an appendix containing the statistical output for the various correlation analyses conducted, including 95% confidence intervals for each of the regression equation parameters.

17. Section 7.2.3 (p. 42) Given EPA's new policy concerning use of IVBA for predicting site-specific bioavailabilty of lead, it would be appropriate to use the IVBA results for the slag storage pile.

18. Section 7.2.4 (p. 43) The equation used to convert IVBA to relative bioavailability (RBA) was revised subsequent to Doe Run's submission of the risk assessment to Region 7. The correct equation is derived in EPA's "Estimation of Relative Bioavailability of Lead in Soil and Soil-Like Materials Using *In Vivo* and *In Vitro* Results" (EPA, 2007a) and is listed below:

$$RBA = 0.878(IVBA) - 0.028$$

This equation should be used to estimate RBA values using IVBA results.

19. Section 7.3 (p. 44) Footnote 13 indicates that Region 7 did not respond to Doe Run's submission of alternative baseline blood lead (PbB) and geometric standard deviation (GSD) levels from the National Health and Nutrition Examination Survey (NHANES 1999-2000 and 2001-2002). As a result, Doe Run ultimately chose to use these values in the draft risk assessment. Region 7 did evaluate Doe Run's proposal, but did not formally respond because EPA was conducting its own analysis of the NHANES data, which recently underwent external peer review. Until EPA completes its analysis and evaluates the policy implications of using alternative blood lead values, the risk assessment should use the PbB

and GSD values from the Midwest Region in EPA's analysis of Phases 1 and 2 of NHANES III (see www.epa.gov/superfund/lead/products/nhanes.pdf). The alternative values used in the draft risk assessment and their potential impacts on predicted blood lead levels should be addressed as part of the uncertainty discussion.

20. **Table 14 (p. 47)** The Adult Lead Methodology (ALM) should not be used to predict blood lead levels for 8 to 10 year old students attending the Taylor School because it is applicable to women of child-bearing age. Rather, Doe Run should use the Integrated Exposure Uptake Biokinetic (IEUBK) model to predict the blood lead levels of 5 and 6 year old children at the Taylor School.

21. **Section 7.3 (p. 48)** The HHRA should clarify how the average inhalation rates for the adolescent trespasser, adolescent recreator, and children at school were derived from the "Exposure Factors Handbook" (EPA, 1997) because it is not readily transparent in the text.

22. **Section 7.4 (p. 49)** (a) This section should briefly explain the batch mode for the IEUBK model and why it is used in this risk assessment. In addition, the text should indicate that a child age of 50 months was chosen because the predicted blood lead level for this age approximates the 6- to 84-month average that is calculated in single run mode. (b) The HHRA also should state that the default dietary lead intake estimates were replaced with updated values using food residue data from the U.S. Food and Drug Administration Total Diet Study and food consumption data from NHANES III (see www.epa.gov/superfund/health/contaminants/lead/ieubkfaq.htm#fda).

23. **Section 8 (p. 51)** As mentioned in the general comments, additional text should be added to the improve the overall transparency of the risk assessment. This section should briefly explain how toxicity assessment is typically performed for both cancer and non-cancer health effects; define toxicity values used in the risk assessment (i.e., reference dose and cancer slope factor); and the process for selecting toxicity values for non-lead COPCs.

24. **Section 8.2 (p. 51)** Doe Run should revise this section to ensure the latest information on the potential adverse health effects of lead are discussed by briefly summarizing the conclusions in the "Air Quality Criteria for Lead" (EPA, 2006), which was developed as part of EPA's reevaluation of the existing National Ambient Air Quality Standard (NAAQS) for lead. This lead criteria document (CD) outlines key findings and conclusions regarding adverse health effects, including neurotoxic effects, cardiovascular effects, renal effects, immune system effects, effects on heme synthesis, effects on bones and teeth, reproductive and developmental effects, and effects on other organ systems. The CD concludes that "...Pb effects occur at blood-Pb even lower than those previously reported for many endpoints (EPA, 2006)."

25. **Section 8.2.1 (p. 51)** The text calls into question whether neurological effects occur below a blood lead level of 10 μg/dL, when in fact there is overwhelming evidence that neurological

effects occur well below 10 μg/dL. The Agency's lead criteria document states "The overall weight of the available evidence provides clear substantiation of neurocognitive decrements being associated in young children with blood-Pb concentrations in the range of 5-10 μg/dL, and possibly somewhat lower (EPA, 2006)." Furthermore, the Agency released its final Staff Paper for the Lead NAAQS on November 1, 2007, which states "In particular, we note that currently available studies provided evidence of adverse health effects associated with blood lead levels and environmental exposures well below those previously identified, and that there is now no discernable threshold for such effects in contrast to the thresholds that had previously been inferred." "As discussed in the CD and summarized in Chapter 3, the current evidence demonstrates the occurrence of a variety of adverse effects, including those on the developing nervous system, associated with blood lead levels extending well below 10 μg/dL to 5 μg/dL and possibly lower." "Further, current evidence does not indicate a threshold for more sensitive health endpoints such adverse effects on the developing nervous system." "In particular, there is now no recognized safe level of Pb in children's blood and studies appear to show adverse effects at mean concurrent blood Pb levels as low as 2 μg/dL (EPA, 2007b)."

These conclusions are supported by the Clean Air Scientific Advisory Committee's (CASAC) review of the CD and Staff Paper, which states "Moreover, there is no evidence of a threshold for the adverse consequences of lead exposure; studies show that the decrements in intellectual (cognitive) functions in children are proportionately greater at PbB concentrations < 10 μg/dL..." "There is also compelling evidence that the risks for mortality from stroke and mycocardial infarction are increased at PbB concentrations below 10 μg/dL, which is considerably lower than those considered acceptable for adults. Finally, although less definitive, there is also evidence that lead exposure during pregnancy is a risk factor for spontaneous abortion or miscarriage at PbB concentrations < 10 μg/dL." "In fact, this evidence suggests these blood lead concentrations below 5 μg/dL are associated with unacceptable adverse effects (Henderson, 2007)."

Last of all, the Centers for Disease Control's Advisory Committee on Childhood Lead Poisoning Prevention recently issued a report stating that "Research conducted since 1991 has strengthened the evidence that children's physical and mental development can be affected at BLLs < 10 μg/dL (CDC, 2007)."

Doe Run should cite these recent evaluations as well as include key conclusions from the documents which clearly show adverse health effects, including neurological effects, at PbB concentrations below 10 μg/dL.

26. Section 8.2.4 (p. 52) The discussion of the carcinogenicity of lead is not consistent with EPA's Integrated Risk Information System (IRIS) which classifies lead as a probable human carcinogen (see www.epa.gov/iris/subst/0277.htm). The Staff Paper (EPA, 2007b) also indicates that both the National Toxicology Program and the International Agency for

Research on Cancer have concluded that lead and lead compounds are probable human carcinogens. Doe Run should delete the current text citing the American Conference of Government Industrial Hygienists (ACGIH) and replace it with appropriate information from IRIS and the Staff Paper.

27. **Section 8.2.5 (p. 53)** As discussed in the comments above, Doe Run should revise this section to ensure the most currently available science is referenced, including the substantial evidence supporting neurological effects in young children with blood lead levels in the range of 5-10 μg/dL and possibly lower.

28. **Section 9.1.2 (p. 55)** (a) In comments dated March 3, 2005, Region 7 requested that the cancer risk for children and adults be added together or an age-adjusted approach be used in the HHRA. The cancer risks should assume an exposure duration of 6 years and 24 years for a child and adult, respectively. Doe Run should revise the exposure assessment text and cancer risk estimates accordingly, as well as the derivation of a preliminary remediation goal for arsenic. (b) The word "COC" should be replaced with "COPC" in this section and throughout the document.

29. **Section 9.2.3 (p. 57)** This section documents that ingestion of cadmium and arsenic in homegrown produce represents a complete exposure pathway. Thus, the HHRA should quantify the potential health risks from this exposure pathway using the sampling results from the Agency for Toxic Substances and Disease Registry (ATSDR) exposure assessment, if the data are adequate.

30. **Section 10.4 (p. 61)** This section should be revised to indicate that only children ages 5 to 7 years old were evaluated at the Taylor School using the IEUBK model (see comment #20).

31. **Section 10.9.1 (p. 65)** If possible, the HHRA should summarize the data on blood lead levels for children living in Herculaneum collected by the Missouri Department of Health and Senior Services (MDHSS) for the last 10 years. This summary should include the number of children sampled, minimum PbB, maximum PbB, geometric mean, number and percentage of children greater than 10 μg/dL.

32. **Section 10.9.1 (p. 66)** Region 7 does not agree that it is standard lead risk assessment practice to compare observed and predicted blood lead levels nor is it appropriate to conduct an empirical comparison on a "broader geographic basis." Empirical comparisons are only appropriate when there is sufficient evidence that the observed blood lead concentrations adequately represent the population and the exposure assumptions in the IEUBK model adequately represent the individual children sampled. In other words, one must ensure that the two populations being compared span similar conditions. It is also important to recall that the IEUBK model is not expected to exactly replicate the observed blood lead concentrations of specific children. Rather, the model is designed to predict the plausible

distribution of PbB concentrations for a child or group of children under a given set of exposure conditions.

As discussed in EPA (1994) and Hogan *et al.* (1998), blood lead data should satisfy several criteria before being used as the basis for comparison to IEUBK model blood lead predictions. For example, paired blood lead and environmental lead levels should be collected within approximately 1 month of each other because the IEUBK model assumes exposure concentrations are relatively constant. Environmental lead concentrations must be characterized in all media (soil, indoor dust, drinking water, air, garden produce, etc.) that contribute to a child's exposure to lead. It is also important to collect behavioral and demographic data, including the time spent away from the primary residence and also to ensure that a child has actually lived at the residence for the 3 months preceding the blood lead measurement. If this type of information is not collected, then an empirical comparison is highly uncertain and one would expect there to be differences between predicted and observed blood lead levels.

It is evident that these criteria have not been satisfied in the Herculaneum risk assessment and, as a result, no conclusions can be reached by this invalid empirical comparison. Therefore, Doe Run should indicate that the data are not adequate to perform an empirical comparison and delete all remaining text which discusses this issue. Rather, the conclusion of this section should state that the existing blood lead data demonstrate there continues to be a significant health threat from lead in this community and that blood lead levels have declined since 1975. This decline is likely due to a variety of factors, including decreases in airborne smelter emissions, residential yard cleanups, and health education.

33. **Section 10.9.2 (p. 67)** The same general considerations regarding adequate exposure characterization apply to comparing predicted blood lead levels using the Adult Lead Methodology and observed blood lead levels in women of child-bearing age. Once again, the empirical comparison is not valid because Doe Run has inadequate exposure information on the adult resident population and the empirical comparison discussion should be deleted. As with young children, the blood lead data indicate that adolescents and adults have been impacted by lead in the community.

34. **Section 11 (p. 69)** Risk based concentrations (RBCs) or preliminary clean-up goals (PRGs) should be derived separately from the risk assessment itself. Thus, Doe Run should move this section to a separate appendix.

35. **Section 11.2 (p. 70)** (a) The PRGs for arsenic and cadmium should be derived using the same exposure parameters used in calculating risks, which includes accounting for the dermal route of exposure. (b) Per the National Contingency Plan, Doe Run should use the "point of departure" or a cancer risk of 1×10^{-6} to derive an arsenic PRG, regardless of whether this value is below naturally-occurring background levels in soil. Region 7 will

ultimately determine the appropriate clean-up level when making a risk management decision for the site. Doe Run should revise the arsenic PRG and the text accordingly.

36. Section 12.1.1 (p. 72) (a) This section cites Dragun and Chiasson (1991) as providing background surface soil concentrations of arsenic and cadmium in Missouri. However, Region 7 previously informed Doe Run that using background surface soil concentrations that are not site-specific values was inadequate and that a statistical hypothesis test should be used to differentiate site-related and background constituents (see "Guidance for Characterizing Background Chemicals in Soil at Superfund Sites" [EPA, 2002]). Because site-specific data are unavailable, Region 7 recommends using the U.S. Geological Survey Pluto Database (see http://tin.er.usgs.gov/pluto/soil/) to characterize the range of background arsenic and cadmium concentrations found in Jefferson County, as well as adjacent counties. If Region 7 determines that remediation is necessary for these two compounds, an appropriate clean-up level will be derived that accounts for naturally-occurring background levels. Doe Run should revise the text accordingly in all sections that reference background levels. (b) Region 7 also does not agree that it is unnecessary to calculate RBCs for arsenic because there are soil concentrations that equate to a Hazard Quotient greater than 1. Doe Run should delete this sentence from the HHRA.

37. Section 12.1.7 (p. 76) In addition to the studies cited in the text, this section should briefly discuss Roberts *et al.* (2007) which evaluated the relative bioavailability (RBA) of 14 soil samples from 12 different sites. The RBA values ranged from 5 to 31% which provides further support for arsenic bioavailability likely being overestimated in the HHRA.

38. Section 12.2.2 (p. 79) The discussion concerning variability of lead concentration as a function of soil particle size should be deleted because Region 7 has recently provided Doe Run site-specific data comparing lead concentrations in the fine (< 250 μm) vs. total soil fractions. Doe Run should evaluate and incorporate these data into the risk assessment.

39. Section 12.2.3 (p. 80) In the fourth sentence, the soil ingestion rate should be revised to 100 mg/day, while the fifth sentence should be revised to 200 mg/day.

40. Section 12.2.4 (p. 81) As requested in previous comments by Region 7, the risk assessment should also acknowledge there is additional uncertainty when using *in vivo* bioavailability estimates for adolescents and adults because evidence exists to indicate that absolute bioavailability of soluble lead (e.g., in food or water) varies with age.

41. Section 12.2.5 (p. 82) Doe Run should provide the output from the regression analysis for the parameters listed in Table 26, including 95% confidence intervals.

42. Section 12.2.5 (p. 83) While Figures 17 to 19 seem to suggest that the IEUBK model default equation underestimates indoor dust lead concentrations, the risk assessment must acknowledge that there is significant uncertainty with this analysis because 26 dust samples

represents only 3% of the properties, the air concentrations are modeled values, and the presence of other lead sources (e.g., lead-based paint, spillage along haul routes, etc.) is unknown. In addition, there is no statistical analysis to support the conclusion that indoor dust lead concentrations decrease with distance from the smelter (see Figure 19). Thus, Doe Run should revise the last sentence in the second paragraph to state "...that the IEUBK model may underestimate the impact...."

43. **Section 12.2.5 (p. 84)** Doe Run should revise the last sentence to state "...the IEUBK model may underestimate...."

44. **Section 12.2.5 (p. 84)** Region 7 does not agree with the conclusion that "...the focus on soil remediation is misplaced...." Rather, the limited data suggest that reducing airborne lead levels should be the highest priority, but lead found in surface soil also significantly contributes to exposure and elevated blood lead levels. Doe Run should delete this paragraph from the risk assessment and the potential impact on clean-up goals should be addressed in the appendix containing the preliminary remediation goals.

45. **Section 12.2.7 (p. 87)** Doe Run should delete both paragraphs on this page referring to Appendix H and replace the appendix with the latest version of EPA's "Lead Soil Trend Analysis" prepared by TetraTech EM Inc., dated August 31, 2007. The text in this section should also be revised to reflect EPA's recontamination analysis contained in Appendix H.

46. **Section 12.2.8 (p. 88)** Doe Run should delete this section from the risk assessment.

47. **Section 13 (p. 90)** (a) The summary should also present the percentage of residential properties in each Exposure Area which exceeds EPA's health protection goal. (b) Doe Run should delete all text which discusses risk-based concentrations.

48. **Section 13 (p. 91)** (a) Doe Run should delete the paragraph discussing observed and predicted blood lead levels, per previous comments on this issue. (b) The primary conclusion of this risk assessment is not that it tends to overestimate risks. Rather, Doe Run must revise the third paragraph to state that the environmental data, blood lead data, and predicted blood lead levels clearly demonstrate there is a significant health threat to young children in Herculaneum.

49. **Section 13 (p. 92)** The last two sentences are Doe Run's opinion concerning how soil clean-up levels should be established by EPA. Doe Run should delete these statements which discuss risk management issues and thus, are not appropriate for the risk assessment.

50. **Tables 16A and 16B** Per comment 28, Doe Run should add another row depicting the total cancer risk for a long-term resident by adding together the adult and child cancer risk estimates.

51. **Figures 9 to 14** The term "*in vitro* bioavailability" should be replaced with "*in vitro* bioaccessibility" because it is technically inaccurate to indicate that *in vitro* models measure bioavailability.

52. **Appendix A** The text should clarify how these modeling results for air and soil deposition were actually used in the risk assessment.

53. **Appendix B** The Data Useability Worksheets are missing information in some fields and should be completely filled out so as to fully address each question.

54. **Appendix D (Tables 2.1 and 2.3)** (a) Per previous comments from Region 7, the "Background Value" column should be deleted and the rationale for COPC detection should be revised from "ABV" to "ASL." (b) Doe Run should delete footnotes 3 and 5 which indicate that background values were used to screen COPCs.

55. **Appendix D (Table 3.1)** The exposure point concentrations for EA 13 and the reference to footnote 4 are missing from this table.

56. **Appendix D (Tables 4.1 and 4.2)** (a) Footnote 3 should be deleted because it is no longer relevant. (b) The reference should be revised to USEPA (2004) in Footnote 4 and in the rest of the document. (c) The grades listed for each school in Footnote 6 should be consistent with the text of the HHRA.

57. **Appendix F** The tables labeled as "Adolescent Lead Model" should be revised to "Adult Lead Methodology" with the words "Adolescent Receptor" inserted below the first line.

References

CDC. 2007. Interpreting and Managing Blood Lead Levels <10 μg/dL in Children and Reducing Childhood Exposures to Lead: Recommendations of CDC's Advisory Committee on Childhood Lead Poisoning Prevention. MMWR 56: No. RR-8.

Henderson, R. 2007. Letter from Dr. Rogene Henderson, Chair, Clean Air Scientific Advisory Committee, to Administrator Stephen L. Johnson. Re: Clean Air Scientific Advisory Committee's (CASAC) Review of the 1st Draft Lead Staff Paper and Draft Lead Exposure and Risk Assessments. March 27, 2007.

Roberts, S.M., Munson, J.W., Lowney, Y.W., and Ruby, M.V. 2007. Relative Oral Bioavailability of Arsenic from Contaminated Soils Measured in Cynomolgus Monkeys. *Toxicol. Sci.* 95(1): 281-288.

U.S. EPA. 1989. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual – Part A. Office of Emergency and Remedial Response, Washington, D.C. EPA/540/1-89/002. December.

U.S. EPA. 1994. Guidance Manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children. Version 0.99d. OSWER Directive No. 9285.7-15-1. Publication No. PB93-963510. Technical Review Workgroup for Lead, Washington, D.C.

U.S. EPA. 1997. Exposure Factors Handbook, Volume I, General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August.

U.S. EPA. 2002. Guidance for Comparing Background and Chemical Concentrations in Soil for CERCLA Sites. Office of Emergency and Remedial Response, Washington, D.C. EPA/540/R-01/003. September.

U.S. EPA. 2004. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment). Office of Solid Waste and Emergency Response, Washington, D.C. EPA/540/R/99/005.

U.S. EPA. 2006. Air Quality Criteria for Lead. Office of Research and Development, Research Triangle Park, N.C. EPA/600/R-5/144aF. October.

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